AMENDMENTS TO THE SPECIFICATION

IN THE SPECIFICATION:

Please amend the paragraph beginning on page 9, line 24, as follows:

"Deficiency in the B5R gene" in the present invention means that the B5R gene is not expressed or that even if the B5R gene is expressed, the expressed protein does not retain any normal functions of the B5R gene product. This deficiency is characterized in that a deleted trait is never reverted to by point mutation in a virus and that reversion to the normal functions that have once been deleted from the B5R gene product will never take place. For example, in the case of the LC16m8 strain, one base has been deleted from the B5R gene by mutation to cause frame shift and ORF of the B5R gene has been shifted, so that normal B5R cannot be expressed. However, if one base is inserted by point mutation that takes place in the vicinity of single-base deletion portion, ORF of the B5R gene is shifted back to the original position. This makes it possible to express B5R having normal functions; that is, to cause atavism. Deficiency in the present invention includes no such deficiency that can cause atavism by point mutation. The B5R gene comprises short consensus sequences included in the region between SCR1 and SCR4 and a transmembrane (TM) domain that plays an important role in the functions of the B5R gene product. Accordingly, "deficiency in the B5R gene" may be deficiency in the transmembrane domain. Moreover, "deficiency in the B5R gene" may be deficiency not only in the transmembrane domain but also in a part of the region between SCR1 and SCR4. In this case, deficiency may be deficiency in the entirety of DNA encoding each region or deficiency such that a part of the DNA encoding each region is deleted, so that no B5R gene products having normal functions will be produced. Moreover, the functional deficiency may be caused by the insertion of the foreign genes into the B5R gene region. Preferably, deficiency in the B5R gene is deficiency in the entirety of the B5R gene or deficiency in the entirety of the transmembrane domain. Furthermore, it is desirable to also delete a promoter in the B5R gene. Such deficiency can be caused by a known homologous recombination method.